UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF PENNSYLVANIA

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION	No. 2:12-md-02323-AB MDL No. 2323
Kevin Turner and Shawn Wooden, *on behalf of themselves and others similarly situated*, Plaintiffs, v. National Football League and NFL Properties, LLC, successor-in-interest to NFL Properties, Inc., Defendants.	Civil Action No. 2:14-cv-00029-AB
THIS DOCUMENT RELATES TO: ALL ACTIONS	

SUPPLEMENTAL DECLARATION OF ROBERT A. STERN, PH.D.

Robert A. Stern, Ph.D., affirms under penalty of perjury the truth of the following facts:

1. I am a Professor of Neurology, Neurosurgery, and Anatomy & Neurobiology at Boston University School of Medicine. I have previously submitted a Declaration (filed 10/6/14) with highlights of my experience, research, and qualifications relevant to the opinions expressed below. I reaffirm the statements set forth in my October 6, 2014 declaration. My complete *curriculum vitae* was attached as Tab A to my October 6, 2014 declaration.

2. My statements and views included in this declaration are mine alone and do not reflect those of Boston University or any of the departments and centers with which I am involved. Specifically, they do not reflect the views of the Boston University Alzheimer's Disease Center, the Boston University CTE Center, or the Boston University Center for the Study of Traumatic Encephalopathy; nor do they reflect the views of any of the faculty, staff, or administration associated with any of these organizations.

3. I have not received any financial payments for preparing this Declaration from any source, including any attorney or plaintiff in this case.

I. CHRONIC TRAUMATIC ENCEPHALOPATHY IS A BRAIN DISEASE

4. CTE is not the same as a brain injury or a concussion. It is a brain disease, accepted as such by the overwhelming majority of thought leaders in the field of neurodegenerative disease. It is described in leading textbooks of Neurology. For example, in "Adams and Victor's Principles of Neurology, 10th edition" (Ropper, Samuels, and Klein; McGraw Hill, New York, 2014) there is an entire section devoted to "Chronic Traumatic Encephalopathy." In yet another important textbook of Neurology, "Merritt's Neurology, 12th edition" (2010, Rowland and Pedley; Lippincott, Philadelphia), there is also a devoted section on "Chronic Traumatic Encephalopathy."

5. The Centers for Disease Control and Prevention (CDC), in its website for the National Institute for Occupational Safety and Health (NIOSH; http://www.cdc.gov/niosh/updates/upd-09-07-12.html, attached as Tab A), includes the following:

> "Other research suggests that chronic traumatic encephalopathy (CTE), a neurological disease that can occur years after exposure to repetitive concussive injuries and exhibits symptoms similar to Alzheimer's disease and ALS in some individuals, has been identified in players who have sustained football-related concussions. The study points out that since CTE is a newly defined diagnosis, it is possible that some deaths attributed to Alzheimer's disease or ALS on death certificates may actually have been related to CTE, though authors were not able to directly assess this in their study."

6. A 2013 "Report to Congress on Traumatic Brain Injury in the United States: Understanding the Public Health Problem among Current and Former Military Personnel" (www.cdc.gov/.../pdf/Report_to_Congress_on_Traumatic_Brain_Injury_2013-a.pdf, attached as Tab B) states: "Multiple severe concussive and sub-concussive injuries, like those reported in boxers who engaged in the sport for several years, are known to cause a delayed dementia syndrome (dementia pugilistica or chronic traumatic

encephalopathy.)" That publication was authored by the CDC and the National Institutes of Health (NIH) in collaboration with the Department of Defense (DOD) and the Department of Veterans Affairs (VA).

7. CTE has been described in the medical and scientific literature since the 1940's. Prior to that time, it was referred to as "Punch Drunk" or "Dementia Pugilistica" when it was believed to occur primarily in boxers. Although the scientific and medical community has known of CTE for almost a century, it is only in the past 10-12 years that it has received more scientific attention. I have stated repeatedly, in professional lectures, in the media, and in scientific journals, that there is much to be learned about CTE. However, the same could be said about other neurodegenerative diseases, such as Alzheimer's disease, ALS, frontotemporal lobar degeneration, and others. That is true even though these other diseases have received more scientific investigation than CTE. In fact, we do not yet know the causes of these other diseases, nor can they be definitively diagnosed during life.

8. CTE has been described in the scientific literature for many decades as involving significant changes in mood and behavior, as well as in cognition. In fact, the changes in mood and behavior are, in large part, what led to the public awareness of this disease in former NFL players.

II. DIAGNOSIS OF CTE DURING LIFE

9. I stated in my October 6, 2014 declaration that a clinical diagnosis of CTE would likely be possible within the next 5 to 10 years, and definitely before the conclusion of the 65-year Settlement term. I reaffirm that position. A clinical diagnosis is a diagnosis of CTE in a living person based on clinical presentation and appropriate objective biological tests or biomarkers. I have reviewed the declarations of Drs. Bernick, DeKosky, Hof, Shenton, Stone, Weiner, Wisniewski, and Zhang, all of whom express the opinion that a "clinically accepted diagnosis of CTE" will likely be possible within the next decade and certainly before the Settlement term expires. I understand that statement to mean, as would those of expertise in the field such as the declarants identified above, that a diagnosis of CTE will be possible in a living person.

10. As I stated in my previous Declaration changes in mood and behavior occur in the general population. Those mood and behavioral changes, however, occur more frequently in individuals with CTE than in the general population. Once a clinical diagnosis of CTE is possible, CTE can be identified as the likely

cause of those mood and/or behavioral changes. Just because there is no currently accepted and validated diagnosis of CTE in the living does not mean a diagnosis will not be possible through the 65-year term of the Settlement.

11. Alzheimer's disease ("AD") is a distinct disease from CTE and also cannot be diagnosed accurately during life. That is, the accurate diagnosis of AD requires a neuropathological examination following death, with the demonstration of excessive depositions of abnormal forms of two proteins, amyloid and tau. However, in recent years, there have been tremendous advances in neuroscience that have improved the accuracy of several tests to diagnose AD during life. One of these tests is a PET scan that is now FDA approved. That scan rules out AD by demonstrating that there is little or no accumulation of amyloid in the brain. There are no FDA approved tests at this time that can positively identify AD as the cause of dementia in the living. However, there are currently clinical trials underway of a new PET scan test to measure and detect abnormal tau in the brain during life. These studies are being conducted on individuals with suspected AD as well as suspected CTE. This test is one of the many promising new technologies that may soon lead to the ability to diagnose CTE in the living.

12. In fact, in addition to the developments and research described in my October 6 Declaration, the National Institutes of Health (through a contribution from the NFL to the Foundation for NIH) will soon be funding a seven-year, $16 million study to "Detect, Define and Measure the Progression of Chronic Traumatic Encephalopathy (http://grants.nih.gov/grants/guide/rfa-files/RFA-NS-14-012.html#sthash.4Oz7Fv5n.dpuf). That request for applications explains that the study is "expected to obtain and use longitudinal data, such as MRI and PET imaging, cognitive and behavioral assessments, and CSF or blood for genomic and proteomic analysis, to increase knowledge concerning the neurological mechanisms of CTE as it evolves over a 3 - 5 year period and enable the development of a consensus, evidence-based clinical diagnosis." It is highly likely that this large NIH-funded project described above will result in an accelerated ability to detect and diagnose CTE during life within the next seven years.

13. It is clear that the advances of neuroscience are at an all-time high, with knowledge and technology leading to exciting new discoveries at an increasingly fast rate. With the advances that have already

taken place in new diagnostic procedures and biomarkers for AD and other neurodegenerative diseases, it is likely, if not assured, that there will be accurate, FDA approved, and clinically accepted methods to diagnose CTE during life over the five to ten years (in large part due to the important new NIH-funded project) and definitely over the 65 years of the proposed Settlement.

III. THE DIAGNOSTIC CATEGORIES OF NEUROCOGNITIVE IMPAIRMENT LEVELS 1, 1.5, AND 2

14. CTE is a neurodegenerative disease. CTE is not a traumatic brain injury nor is it a concussion. It is a progressive brain disease that begins early in life. As it destroys more and more brain tissue, it leads to a variety of symptoms, including mood and behavioral symptoms as well as cognitive decline. The examination of individuals with neurodegenerative diseases, such as CTE and AD, requires special training, experience, and expertise. The development of criteria to classify the level of neurocognitive impairment associated with CTE requires knowledge of and expertise in the clinical presentation of neurodegenerative disease, including dementia. The classification scheme of Neurocognitive Impairment 1, 1.5, and 2 included in the proposed Settlement, does not exist anywhere in the neurodegenerative disease or dementia literature.

15. A diagnostic classification scheme for neurocognitive impairment, especially one that is used for the purpose of financial compensation, must undergo extensive validation studies, specifically in the population being examined – here, the population of former NFL players . This process normally takes several years to complete. The algorithm included in the proposed Settlement has not undergone any validation study. Although each individual test within the battery has been scientifically validated, the test battery in its entirety, and most importantly, the specific algorithm used to define Neurocognitive Impairment categories, has not been validated in any population, much less the population of individuals who will be the classified in this proposed Settlement, i.e., former NFL players.

16. According to Dr. Scott Millis' Declaration, (Dkt. No. 6422-34) the "algorithm was based on extensive research performed by Dr. Grant Iverson—one of Class Counsel's consultants." He cites three publications to support this statement, referring to them as "studies" (Brian L. Brooks et al., *Developments in Neuropsychological Assessment: Refining Psychometric and Clinical Interpretive Methods*, 50 Canadian Psychol. 196, 2009, "Brooks Study"; Brooks, B. & Holdnack, J., *Evidence-Based Neuropsychological*

Assessment Following Work-Related Injury, *in Neuropsychological Assessment of Work-Related Injury* 360, 360-400; in Shane S. Bush & Grant L. Iverson eds., 2012; "Holdnack Study"; and Grant L. Iverson & Brian L. Brooks, *Improving Accuracy for Identifying Cognitive Impairment*, *in The Little Black Book of Neuropsychology: A Syndrome-Based Approach* 923, 923-950; in Mike R. Schoenberg & James G. Scott eds., 2011; "Iverson Study"). Two of these publications are chapters in books and are not peer reviewed. The third publication (Brooks et al., 2009) is in a psychology journal but is a review article and not a research study. I have reviewed these publications and none focuses on the evaluation of older individuals with dementia and none of these publications provides a description of the algorithm used in the proposed Settlement.

17. The individuals who authored these publications are well-respected in the field of neuropsychological assessment and psychometrics. In fact, I have published a chapter in a Neuropsychology Handbook with Drs. Iverson and Brooks about the *Neuropsychological Assessment Battery* (NAB), a battery of 33 neurocognitive tests that I created (with Dr. Travis White), funded, in part, by the NIH, and which took 7 ½ years to develop. A review of each of the primary authors' (Holdnack, Brooks, Iverson) online biographies, however, shows that they do not specialize in dementia or neurodegenerative disease.

IV. CO-LEAD CLASS COUNSEL SELECTIVELY QUOTED MY WRITINGS AT THE FAIRNESS HEARING

18. I attended the November 19, 2014 fairness hearing and listened to the arguments of Class Counsel, Counsel for the NFL Parties, and various counsel for Objectors. I have also reviewed the relevant portions of the transcript from that hearing.

19. During his rebuttal argument, Mr. Seeger quoted from several of my scholarly publications and suggested that my academic writings undermine or contradict the opinions I expressed in my October 6 Declaration. The opinions I expressed in my October 6 declaration are entirely consistent with my body of academic research.

20. First, Mr. Seeger quoted me as writing that "there is no epidemiological cross-sectional prospective studies of CTE that currently exist." I did not, however, suggest otherwise in my October 6 Declaration. In focusing on the absence of prospective studies, Mr. Seeger ignores the significance of the retrospective studies that have been performed. Those studies have generated a consensus in the scientific

community that repetitive head trauma is a necessary condition for developing CTE. That is, in the absence of repetitive head trauma, an individual will not develop CTE. Since the 1970s, every case of neuropathologically confirmed CTE (nearly 200) has been found in an individual who sustained repetitive head trauma. No case of neuropathologically confirmed CTE has yet been identified in an individual who has not sustained repetitive head trauma.

21. Second, Mr. Seeger quotes a 2014 article of mine that states: "There are no objective validated *in vivo* biomarkers of CTE." Again, my October 6 declaration does not state otherwise. Rather, I noted that tests for such biomarkers would likely be available within 5 to 10 years, and that those tests could be combined with clinical examination to produce a highly accurate, clinically accepted, and FDA-approved method of diagnosing CTE during life.

22. Finally, Mr. Seeger quotes me as writing that "although a history of remote head trauma may be suggested of CTE, head trauma has been implicated as a risk factor for Alzheimer's, Parkinson's disease, ALS and other neurodegenerative diseases." Even though head trauma has been implicated as a risk factor for Alzheimer's, Parkinson's, ALS, and other neurodegenerative diseases, it is ***also*** a risk factor for CTE. And, importantly, of the neurodegenerative diseases for which head trauma is a risk factor, CTE is the ***only*** disease that requires head trauma as a necessary condition. In contrast to CTE, each of the other neurodegenerative diseases is found in individuals who have not suffered repetitive brain trauma.

Pursuant to 28 U.S.C. § 1746, I state under penalty of perjury that the foregoing is true and correct:

Robert A. Stern, Ph.D.

Date: November 30, 2014